Review Article

Echocardiographic Evaluation of a Patient in Circulatory Shock: A Contemporary Approach

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Abstract

Circulatory shock is characterized by a state of inefficient tissue oxygen supply and multiple organ dysfunction. Patients with circulatory shock require fast and assertive diagnosis and therapies to reduce its high lethality. Echocardiography has already been established as a fundamental method in managing patients with circulatory shock. It provides crucial assistance in etiological diagnosis, prognosis, hemodynamic monitoring, and volume estimation in these patients; its potential advantages include portability, absence of contrast or radiation, low cost, and real-time serial assessment. In the intensive care unit setting, it demonstrates a high correlation with invasive (pulmonary artery catheter) and minimally invasive (transpulmonary thermodilution) forms of hemodynamic monitoring. Currently, other techniques, such as pulmonary ultrasound and VExUS score, have been added to echocardiographic assessment, making the method more comprehensive and accurate. These techniques add relevant data to blood volume estimation in critical patients, influencing the probabilistic decision of fluid responsiveness and providing additional information in the diagnostic reasoning of the causes of shock, thus optimizing these patients' prognosis. Point of care ultrasound (POCUS) aims to make abilities to obtain information at the bedside more accessible to physicians who are not specialists in radiology, by means of ultrasound, which assists them in decision-making. This article addresses the diverse applications of echocardiography in patients with circulatory shock, including prognostic evaluation and etiological diagnosis by means of the parameters found in the main causes of shock, in addition to hemodynamic monitoring, evaluation of fluid responsiveness, and practical use of pulmonary ultrasound.

Introduction

The use of the echocardiogram is becoming more common in the daily practice of the intensive care unit (ICU), thus bringing several forms of application in the handling of the critical patient. Besides the many types of information that can be obtained by the examination, there are advantages brought by the method itself, such as portability, lack of contrast or radiation, low cost and serial evaluation in real-time.[1]

Circulatory shock is characterized by a state of inefficiency of the tissue oxygen offer, leading to cell failure and multiple organ dysfunction. Therefore, patients with such a condition require fast and assertive diagnosis and therapies for the reduction of its high lethality. The point of care ultrasound (POCUS) aims at making the skills to obtain bedside information more accessible for the physician that is not specialized in radiology, through the ultrasound, helping them in the decision-making process.[2] Complementation with a comprehensive echocardiography, performed by an imaging expert, makes this relationship even narrower and beneficial, optimizing the prognosis of the critical patient.

In this article, we describe several forms of application of the echocardiogram and POCUS in patients in circulatory shock, including diagnosis, prognosis, hemodynamic monitoring, fluid-responsiveness evaluation and use of a lung ultrasound.

Keywords

Circulatory shock; Diagnosis; Prognosis; Lung ultrasound; Fluid-responsiveness.

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Echocardiographic evaluation by etiology

Cardiogenic shock

Cardiogenic shock is caused by a severe involvement of the myocardial function, leading to reduced cardiac output, tissue hypoperfusion and hypoxia. In patients hospitalized with acute heart failure (HF), the incidence of cardiogenic shock is of about 12%, and ischemic disease is responsible for approximately 80% of these cases. [3] Despite the significant advances in reperfusion therapy and circulatory support devices, mortality rates remain high, reaching up to 50%.[4]

Due to the multiplicity of causes and physiopathological mechanisms that lead to cardiogenic shock, its recognition is based on the combination of low cardiac output, signs of high filling pressure and structural and functional heart disease. The typical pattern includes low systolic volume associated with high left atrial pressure (increased mitral flow E-wave velocity and E/e' ratio).[5] The left atrium (LA) and left ventricle (LV) size may provide clues about the duration of the contractile involvement, and dilation indicates a stage of chronicity.

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The LV ejection fraction (LVEF) is a traditional and practical parameter, besides a useful bedside guide to estimate systolic function. The interpretation needs to consider the preload effects (volemia), blood pressure (afterload), inotropics and vasopressors. The estimation of the LV systolic volume and cardiac output through the echocardiogram is well validated, being a useful parameter for the diagnosis of cardiogenic shock. The velocity time integral (VTI) of the left ventricular outflow tract (LVOT) can be used as a replacement for systolic volume, being a more practical parameter to be obtained, with lower interobserver variability. If it is higher than 18 cm, it is suggestive of proper systolic volume.[6]

In cardiogenic shock caused by ischemia, it is important to assess the associated mechanical complications: acute mitral insufficiency (MI), rupture of the interventricular septum (IVS) or the myocardial wall with or without formation of a pseudoaneurysm. All of these complications usually have a bimodal peak incidence.[7]

Cardiogenic shock associated with isolated right HF is characterized by low cardiac output associated with high right atrial pressure, dilated right atrium (RA) and dilated inferior (IVC) and superior vena cava, without significant pulmonary hypertension (PH), besides the finding of a dilated right ventricle (RV) with dysfunction. It is associated with inferior wall acute myocardial infarction (AMI), with proximal occlusion of the right coronary artery, and it is an adverse prognostic marker.[8]

Valvulopathy is a major cause of cardiogenic shock. The echocardiogram plays an essential role at identifying valvular disease, its severity and possible complications. Atrial fibrillation with high ventricular response in patients with mitral stenosis, significant valvular regurgitation due to infectious endocarditis or degenerative disease (chordae tendineae rupture) or aortic failure secondary to aortic dissection are some examples of acute causes, which lead to a challenge in the echocardiographic evaluation. Severe aortic stenosis can also be presented in cardiogenic shock, but it is usually secondary to acute decompensation in the context of subjacent severe chronic systolic dysfunction.[4]

There are many other conditions, such as stress cardiomyopathy, acute myocarditis, tachycardiomyopathy, which can be presented as cardiogenic shock. In these cases, the echocardiogram will show a compromised LV systolic function, with reduction of the VTI of the LVOT, suggesting the cardiogenic origin of the shock. The systolic dysfunction of the LV is usually characterized by diffuse hypokinesis, except for cardiopathies that can lead to segmental changes in contractility, such as ischemic cardiopathy or Takotsubo cardiomyopathy. Regional involvement is characteristic of the latter, showing apical ballooning and hypercontractility of basal segments.[9]

Acute pulmonary thromboembolism (APT)

In acute pulmonary embolism, there is a sudden increase in pulmonary vascular resistance (PVR), overlapping the capacity of the RV, which is adapted to a low-pressure regime. Consequently, there is acute dilation in the basal and medium regions of the RV, associated with a compromised systolic function. There is some preservation of the apical region of the RV, limited by the insertion of a lateral moderator band, in which systolic function is normal or increased. This finding, called "McConnell's sign",[10] is characteristic, but not pathognomonic, also occurring in RV infarction.

The RV prolongs its systolic time when faced with an afterload increase, so that the pressure in the RV exceeds the pressure in the LV at the end of the systole; such an effect is exacerbated by the underfilling of the left heart due to pulmonary oligoemia. Therefore, we find the paradoxical motion of the IVS with systolic rectification (observed at the parasternal short axis view when the LV takes on a D-shape during systole). The dilation of the RV is the keystone, and its association with the paradoxical septal motion defines the acute *cor pulmonale*. The dilation of the RV is promptly evaluated in the apical four chamber view, when the RV/LF ratio is >0.6; severe dilation is seen with a >1.0 proportion.[11] Acutely, the diameter of the pulmonary artery and the volume of the RA remain normal.

The evaluation of the LV is also informative in massive pulmonary thromboembolism (PTE), with small cavity and reduction of cardiac output. The presence of thrombus in the right chambers, IVC or pulmonary artery is occasionally seen, and reinforces PTE as a shock etiology – characterized as a low-flow obstructive shock, when VTI of the LVOT and cardiac output are typically low.[12]

The measures of the pulmonary artery systolic pressure (PASP), systolic function of the RV and PVR are useful to confirm the increased afterload of the RV, and especially to assess the effects of thrombolysis and other interventions.

• PASP is more commonly obtained through the peak tricuspid regurgitation velocity, using the modified Bernoulli equation. It is important to pay attention to the alignment and obtaining of accurate Doppler signals to prevent the underestimation of measures. In the absence of a reliable signal of tricuspid regurgitation, the pulmonary ejection acceleration time (PAAT) can be used. As a reference, a 70-90 ms PAAT indicates PASP > 70 mmHg. The presence of a mid-systolic notch at the pulse Doppler in the right ventricular outflow tract (RVOT) also indicates severe pulmonary arterial hypertension (PAH).[13] In acute pulmonary embolism with hemodynamic repercussion, we typically see PASP with mild to moderate increase.

• The systolic function of the RV can be normal, hyperdynamic, right after the pulmonary embolism insult, or hypodynamic in further stages. The tricuspid annular plane systolic excursion (TAPSE) is an acceptable and easy method to be used at the bedside, with a narrow learning curve (normal value ≥ 17 mm). The velocity of the lateral tricuspid annular plane at the tissue Doppler can also be used, and the value of < 9.5 cm/s means ventricular dysfunction. More accurate parameters should be used whenever possible, such as: variation of the fractional area (normal value ≥ 35%), right ventricular free wall longitudinal strain (normal value > 20%), and ejection fraction in the 3D echocardiogram (normal value ≥ 45%);

however, these are not always feasible for the critical ICU patient at bedside.[14]

• PVR: the gold standard for PVR is right heart catheterization; however, several methods have been proposed to estimate PVR using the echocardiogram, each one using the principle of pressure in relation to cardiac output (that is, the ratio between the pressure in the pulmonary arterial bed and cardiac output). The following formula, proposed by Abbas and collaborators, can be used:[15] PVR = TRV / VTI_{RVOT} x 10 + 0.16, in which TRV is the tricuspid regurgitation velocity, which corresponds to the transpulmonary pressure gradient, and VTI_{RVOT} is the VTI of the RV outflow tract, which corresponds to pulmonary blood flow (PBF). When compared to the PVR estimated by hemodynamics, the author obtained excellent correlation, R = 0.929. However, this calculation can be influenced by other factors, such as the variation of the alveolar pressure and pulmonary venous pressure: the increased pressure in the LA reduces PVR.

Therefore, in practical terms, the echocardiographic findings of RV dilation, paradoxical septal motion and McConnell sign suggest the diagnosis of PTE with obstructive shock as a cause, and the findings of elevated PSAP and PVR, together with reduced cardiac outflow and systolic function of the RV, support it, being an essential tool in the indication of thrombolysis.

Sepsis

Sepsis is the most prevalent cause of shock in an ICU, and it is closely related to cardiac injury in severe cases. In practice, septic shock has a distribution pattern, and is characterized in the echocardiogram by the presence of hyperdynamic ventricles and high cardiac output (normal or high VTI in RVOT associated with tachycardia).[16] However, the evolution with concomitant ventricular dysfunction is observed in 20-60% of the severe cases and is associated with worse outcomes.[17]

A major pathological contribution for shock in sepsis is peripheral vasoplegia. Systemic vascular resistance (SVR) can be calculated using the formula: SVR = (mean arterial pressure - ventral venous pressure / cardiac output) x 79.9 (dyn.s/cm^{-5}), and is typically reduced in these patients. Besides, hypovolemia is frequently associated as a consequence of the reduced effective circulating volume related to venous dilation and to the increased capillary permeability with losses for the third space. Heart dysfunction is mainly caused by the liberation of cytokines, mitochondrial dysfunction and tissue hypoxia, which lead to myocardial lesion.[17]

The spectrum of involvement in septic cardiomyopathy can range, including dysfunction in the LV and/or RV, global hypokinesia or changes in segmental contractility and subtle findings only identified with strain.[18] The diastolic dysfunction of the LV with high filling pressure is also very common in this scenario. A recent meta-analysis showed reduced e' wave associations and high E/e' ratio as predictors of higher mortality rates among critical patients with sepsis.[19]

Many patients with myocardial depression in sepsis do not require the use of inotropic agents, because cardiac output is adequate even at the presence of damaged cardiac function, due to the associated reduction of the LV afterload and its mild dilation.

It is important to highlight that contractile dysfunction is almost always reversible throughout the days, except in the presence of concomitant subjacent coronary artery disease or myocarditis. The reduction in the LV afterload may mask systolic dysfunction, which can become obvious only after the correction of hypotension.[20]

The echocardiogram plays a pertinent role in the evaluation of the valves in septic shock. Endocarditis or perivalvular abscesses can be the cause of shock. Transesophageal echocardiogram (TEE) is the technique of choice in the presence of such a suspicion, even though the transthoracic echocardiogram is still valuable in the acute scenario.

The RV can also be impacted by the combined effects of sepsis in contractility and higher RV afterload (acute respiratory distress syndrome [ARDS] and mechanical ventilation). In 20% of the patients, RV dysfunction is the prevalent characteristic.[21]

Finally, it is important to verify the presence of dynamic outflow tract and mid-ventricular obstructions in the LV, once the combination of the hyperdynamic state and hypovolemia can induce them and contribute with the genesis of hypotension.

Therefore, the bedside echocardiogram in patients with septic shock provides valuable information and can help with strategies to handle this context.

Cardiac tamponade

Pericardial effusion is the accumulation of fluid in the pericardial space beyond the physiological volume. It is especially related to inflammatory causes, post-operative periods of heart surgery and as a complication of percutaneous heart procedures. Cardiac tamponade is a clinical diagnosis that happens when the pressure made by the pericardial fluid overcomes the pressure in the heart chambers. The classical signs of the Beck's triad (hypotension, jugular venous distension and muffled heart sounds) have limited sensitivity, turning clinical diagnosis into a challenging task.[22]

The echocardiogram plays an essential role in the quantification of pericardial effusion, definition of the etiological suspicion and its repercussion in ventricular filling, especially in the finding of early changes that precede the clinical tamponade. In the physiopathology of cardiac tamponade, the velocity of the installation of the effusion plays an essential part, beyond its magnitude. This information becomes relevant in scenarios in which the fast installation of the small pericardial effusion (right ventricular free wall rupture after the infarction, hemopericardium as a complication during percutaneous heart procedures) is sufficient to determine hemodynamic involvement. The volemic status of the patient also contributes with

the occurrence of clinical repercussion, and this data is also accessible in a non-invasive manner, through the echocardiogram. Besides, the echocardiogram can be essential in urgent situations, to guide the subxiphoid puncture for pericardiocentesis (Marfan's puncture).

The first step in the diagnosis of pericardial effusion is to differentiate it from the pleural effusion. In the parasternal long axis window, pericardial effusion is in an anterior position in relation to the descending thoracic aorta, in a cross-sectional view; the pleural effusion is posterior to that structure (Figure 1). It is important to differentiate the epicardial fat from the pericardial effusion, which has lower echogenicity in comparison with the myocardium, and moves along with it.

The quantification of the pericardial effusion is based on the size of the layer of fluid measured in diastole. If the layer is smaller than 10 mm, the effusion is considered as mild; between 10-20 mm, moderate; and higher than 20 mm, it is a major pericardial effusion.[23] It becomes a challenge to accurately quantify and grade the effusion when its distribution is heterogeneous.

The presence of debris, mass or clots should also be reported in the exam's report, contributing with the etiological diagnosis and proper treatment.

In the patient with hemodynamic instability associated with pericardial effusion, some objective data from the M-mode analysis and bidimensional method, besides changes in blood flow by the Doppler, are able to determine the presence of restriction in ventricular filling.[23]

The following findings can be observed in patients with cardiac tamponade (Figure 1):

a. Paradoxical motion of the IVS: bulging of the IVS towards the LV during inspiration. Echocardiographic representation of ventricular interdependence, being the mechanism in charge of the pulsus paradoxus that is present in tamponade.

b. Diastolic collapse of the RA: sensitive and early finding because it is a low-pressure chamber. This finding has more value when the duration of the RA collapse is superior to one third of the cardiac cycle.

c. Diastolic collapse of the RV: less sensitive finding, however, more specific for the tamponade diagnosis.

d. Respiratory variation of the mitral flow E-wave (> 25%) and of the tricuspid flow E-wave (> 40%).

e. Reduction of systolic volume and cardiac output during inspiration (echocardiographic representation of the pulsus paradoxus).

f. Dilated inferior vena cava (IVC > 21 mm) associated with respiratory variation lower than 50%. This is a very sensitive finding in the tamponade diagnosis, however, little specific, since it can be present in other clinical conditions.

Hypovolemia

The echocardiogram may provide several objective parameters of hypovolemia, which help in the etiological diagnosis of shock and in the monitoring of the response after volemic repositioning.

In severe hypovolemia we can see the collapse of the LV at the end of systole, sign that is classically called "kissing walls". However, this finding is not specific for the diagnosis of hypovolemia, since it can be present in conditions of low SVR, hyperdynamic states with high cardiac output or use of inotropics. Among hypovolemic patients, it is also possible to find reduced final diastolic diameter of the LV, inferring low preload. On the other hand, the fixed bulging of the interatrial septum addressed to the RA suggests increased left atrial pressure and possible reestablishment of normovolemia.[24]

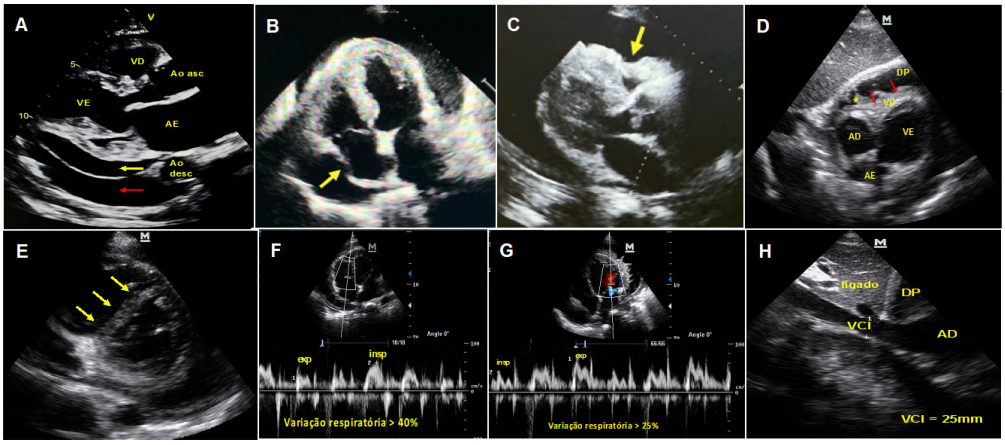

Figure 1 – *Evaluation of pericardial effusion and signs of cardiac tamponade. A. Differentiation between pericardial effusion (anterior to the descending aorta – yellow arrow) and pleural effusion (posterior to the descending aorta – red arrow). B. Transient collapse of the right atrium (arrow). C and D. Diastolic collapse of the right ventricle (arrow). There is presence of debris (asterisk). E. Paradoxical movement of the interventricular septum – displacement of interventricular septum towards the left ventricle during inspiration (echocardiographic representation of ventricular interdependence). F and G. Significant respiratory variation of E-waves of the mitral and tricuspid flow. H. Subcostal window showing plethoric inferior vena cava.*

Another parameter that is usually found among hypovolemic patients is collapsed IVC or with reduced dimensions. It is important to remember that in situations of right ventricular dysfunction, significant pericardial effusion, increased intra-abdominal pressure and in patients on mechanical ventilation, the use of IVC diameter and its respiratory variation becomes less reliable.[24]

Other common findings, however, not specific of hypovolemic shock, are VTI of LVOT below 18 cm, low systolic volume, low cardiac output and high SVR. One of the ways to distinguish it from a cardiogenic shock is to assess the filling pressure through the E/e' ratio, which will be reduced (E/e' < 8) in hypovolemic shock, besides the absence of dilation or LV dysfunction.[25]

The echocardiogram in hemodynamic monitoring

As observed, the echocardiogram can provide, in a non-invasive and serial manner, hemodynamic parameters using data from the Doppler and measures from the bidimensional method. In Figure 2, we describe how to classify the patient in circulatory shock profiles using echocardiographic parameters.

Besides showing high correlation with invasive (pulmonary artery catheterism) and minimally invasive forms (transpulmonary thermodilution) of hemodynamic monitoring, the echocardiogram presents the advantage of directly visualizing the heart structures and their functionalities.[24] Nowadays, other techniques, such as pulmonary ultrasound (PUS) and the VExUS score, have been added to the echocardiographic evaluation, making the method more comprehensive and accurate.[26,27]

The E/e' ratio, parameter that is exclusively obtained by the echocardiogram, has high correlation with the filling pressure of the left heart chambers, with significant prognostic value in the clinical context of the critical patient. Several studies have demonstrated that the E/e' ratio > 15 is predictive of an unfavorable outcome.[28]

The ventricular-arterial coupling (VAC) has become popular in the evaluation of the patient with cardiopathy. It is defined as the inter-relation between the left ventricular function and the arterial system, thus reflecting the global cardiovascular performance.[29] Mathematically, VAC is defined by the arterial elastance (Ea) divided by ventricular elastance at the end of the systole (Ees). Deriving from the Chen formula, the non-invasive measure of Ea and Ees is taken using simple data, such as systolic blood pressure, diastolic blood pressure, systolic volume, ejection fraction, time of total ejection and pre-ejection time. The normal values considered in consensus are: Ea = 2.2 ±0.8 mmHg/ml and Ees = 2.3 ±1.0 mmHg/ml. VAC (Ea/Ees) = 1 represents the ideal coupling, when there is the best ventricular work. Ea/Ees > 1 represents the ventricular-arterial decoupling, demonstrating inefficiency of the ventricular work. In the patient with decompensated HF, we can use VAC to guide therapy. When VAC >1 due to expressive increase in Ea, the therapy with vasodilators would reduces the Ea and, consequently, VAC. In the case of VAC > 1 due to reduction of Ees, therapy with inotropics would bring the ratio closer to the ideal value of 1.[30] As to the patient with septic shock, it is usual to see ventricular-arterial decoupling, especially due to expressive reduction of Ees. In this case, therapy with fluids and inotropics would restore the Ea/Ees ratio. On the other hand, the use of vasopressors alone would lead to worse coupling due to the isolated increase of Ea.[31]

Figure 3 shows how to measure the main echocardiographic parameters and their respective formulas.

Evaluation of fluid responsiveness

In-water resuscitation of the patient with circulatory shock aims at incrementing systolic volume and, consequently, cardiac output to improve tissue oxygenation. However, about half of the patients are considered as non-responsive to fluid therapy, and the early identification of this profile may prevent the risk of water overload.[32] Several parameters have been assessed as echocardiographic predictors of response to fluids. IVC is one of the classic parameters in the composition of the fluid responsiveness evaluation, and its plausibility is based on the lung-heart interaction. The variation of transpulmonary pressure while breathing is transmitted to the right cavities, making the venous return

SHOCK	IVC (RAP/CVP)	E/e' (PCP)	CO / CI	SVR	PVR
Hypovolemic	↓	↓	↓	↑	↓
Cardiogenic	↑	↑	↓	↑	↓
Distributive (septic)	↓	↓	↑↓	↓	↓
Pulmonary thromboembolism	↑	↑↓	↓	↑	↑
Cardiac tamponade	↑	↑↓	↓	↑	↓

Figure 2 – *Behavior of echocardiographic parameters according to type of shock. IVC: inferior vena cava; RAP: right atrium pressure; CVP: central venous pressure; PCP: pulmonary capillary pressure; CO: cardiac output; CI: cardiac index; SVR: systemic vascular resistance; PVR: pulmonary vascular resistance.*

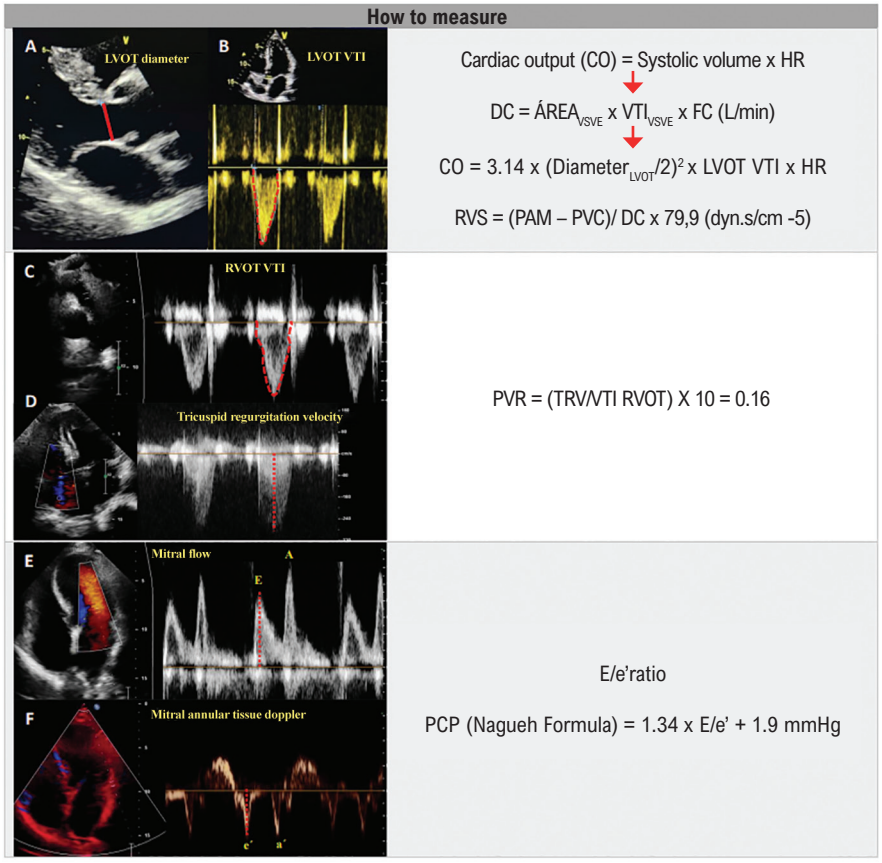

Figure 3 – *How to measure hemodynamic parameters with the echocardiogram. A and B: Cardiac output (CO) calculation, using the LVOT diameter (A) and LVOT VTI (B). Calculation of systemic vascular resistance, using CO, MAP and CVP estimation by the collapsibility index of the inferior vena cava; C and D: Calculation of pulmonary vascular resistance, using RVOT VTI (C) and tricuspid regurgitation velocity (D); E and F: Calculation of the E/e' ratio, using the E-wave mitral flow velocity (E) and the e'wave derived from the lateral mitral annular tissue Doppler (F). Calculation of PCP, using the E'e ratio by the Nagueh formula. CO: cardiac output; HR: heart rate; LVOT: left ventricle outflow tract; VTI: velocity time integral; SVR: systemic vascular resistance; PVS: pulmonary vascular resistance; TRV: tricuspid regurgitation velocity; RVOT: right ventricle outflow tract; PCP: pulmonary capillary pressure.*

vary. This ratio is influenced by the ventilatory mode and the complacence of the vena cava.[33]

IVC

The current recommendations suggest the evaluation of IVC to estimate the pressure inside the RA in non-ventilated patients, due to collapsing during inspiration: IVC diameter < 21 mm and collapsing > 50% suggests normal pressure (between 0 and 5 mmHg), whereas diameter > 21 mm and collapsing < 50% suggests high pressure (between 10 and 15 mmHg).(32) Vena cava diameter < 10 mm is common in states of hypovolemia and may indicate higher probability of response to fluids, whereas a diameter higher than 25 mm is associated with high volemic status and low probability of response to fluids. However, these static values are limited in the prediction of response to fluids, and the dynamic method based on the variation of IVC while breathing (distensibility index) allows to assess fluid-responsiveness more accurately. This calculation can be made using two different formulas, using the diameter of IVC at the end of inspiration and the end of expiration.[34] In ICUs with non-ventilated patients, the studies show high specificity, but low sensitivity.

Variation of systolic volume

The variation in systolic volume is a good indicator of fluid responsiveness, being easy to acquire and reproduce.[35] A variation in systolic volume higher than 12% was accurate to predict fluid responsiveness with values higher than 14%, being a strong predictor, and values below 10% with low correlation and response to volemic repositioning.

Even though static parameters have a limited value in the prediction of responsiveness to volume, such parameters can provide additional information for the probability evaluation in bedside decisions regarding resuscitation with fluids in the circulatory shock.

Cavity dimension

The presence of reduced LV dimensions with final diastolic area of the LV in the cross-sectional axis at the level of papillary muscles < 10 cm² with signs of hypercontractility (*"kissing walls"*) is a strong predictor of hypovolemia.[33]

The RV dilation after massive administration of fluids is usually seen in patients in volemic resuscitation, and it may work as one of the parameters for the adoption of protective ventilatory measures and interruption of the strategy of fluid administration.

Passive *leg raising* (leg elevation test)

A simple method used at bedside that uses the elevation of lower limbs at 45º and parameters of systolic volume or isolated VTI with variation above 10% suggests fluid responsiveness.[33] Echocardiogram remains as a practical instrument and with a valuable additional resource in the decision of administering fluids in the resuscitation of patients with acute circulatory failure, aiming at incrementing the systolic volume, and, consequently, cardiac output.

VExUS Ultrasound

The presence of right heart chamber overload, determining systemic venous congestion, has been underestimated in the evaluation at bedside of the critical patient due to the limitation of the traditional physical exam. The development of a tool addressed to systemic venous ultrasound (VExUS) and the growth of the *"point of care"* in the past decade enabled the use of a useful tool in the intensive care practice.[26]

Recent data guide the role of the VExUS score ultrasound for the evaluation of systemic congestion through the assessment of VCI and the venous flow of hepatic, renal and portal veins (Figure 4).

Under normal circumstances, the venous compartment is highly complacent with high capacitance; therefore, the more distal the heart, the more blunted the venous pulse, so that in smaller veins the flow becomes wavy, with a phasic nature. However, in states of right ventricular failure or overload in intravascular volume, the venous compartment is congested, and the limits of venous complacence are reached. Under these circumstances, the normal blunting of the venous pulse, due to the complacent nature of the smaller veins, is lost, and the pulsations are transmitted in a retrograde manner to the smaller veins.[36]

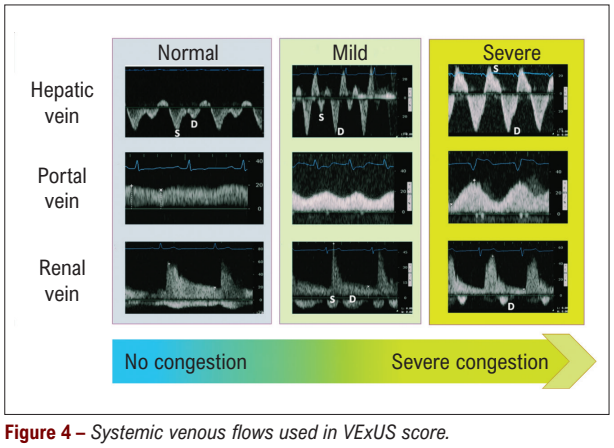

Figure 4 – *Systemic venous flows used in VExUS score.*

Hepatic venous circulation follows this path: portal vein, hepatic sinusoids, hepatic veins and IVC. Due to the distance from the heart, the normal portal venous flow is wavy and has a phasic nature. While the venous congestion increases, the retrograde flow of the hepatic vein generated by atrial contraction is transmitted by hepatic sinusoids and to the portal vein, where the impedance to the hepatoportal flow begins. This makes the normal wavy flow become progressively pulsatile, and this phenomenon is aggravated when there is systolic reversion of the venous return to the heart. Eventually, pulsatility becomes significant enough to cause a biphasic pattern. The intrarenal Doppler signal is usually a continuous monophasic flow below the baseline. With the increasing venous congestion, venous flow becomes pulsatile, and then progresses to an interrupted biphasic flow that is correlated with the S and D waves of the hepatic vein flow.[36]

PUS in shock

The use of a PUS has been highlighted in several clinical scenarios, and especially in critical intensive care patients. In patients with shock, it has a huge potential of helping the etiological diagnosis, besides providing information related to the level of lung involvement, associated congestion and/or complications related to mechanical ventilation.[37] Its feasibility at bedside, low cost, absence of radiation and obtention of this information in a serial manner are, without question, advantages of this method in relation to the others.

Technical aspects of PUS

PUS may require sectoral, convex or linear probes. Due to the physical principles inherent to the method, those transducers with lower frequency (sectoral and convex) will be ideal to assess lung parenchyma, whereas those with higher frequency will better visualize the subpleural and pleural space.[38] We should sweep using the ultrasound in several zones, and the disposition of the transducer can be perpendicular to the ribs, as well as along the intercostal spaces.

PUS can be performed both in the sitting and in the supine position; however, the position should be constant, because pulmonary fluid changes according to posture, and this must be written in the report. The device should be configured for the better processing of the image, adjusting frequency, depth, focus and gain appropriately. Each point should be assessed for at least one complete respiratory cycle.[38]

Many protocols have been validated in the literature, with the thoracic wall being divided in quadrants and the changes reported by sectors, or as a sum of each

region.[39] A more comprehensive and systematic approach is recommended whenever possible, dividing the thorax in 12 (upper and lower portion in the anterior, lateral and posterior region of each hemithorax), or 8 quadrants (excluding the posterior region). However, in emergency situations, requiring fast bedside answers, we can use a more focused and simple analysis. In this sense, a total of 6 quadrants is recommended: anterior, upper and posterolateral of each hemithorax.

Applicability of the PUS

The normal PUS image is characterized by lung sliding and by repetitions of hyperechogenic horizontal lines, called A Lines. The reduction in lung aeration due to inflammatory alveolar infiltration or congestion triggers the onset of vertical hyperechogenic lines, called B lines.[40]

PUS is more sensitive to detect pulmonary congestion when compared to the clinical exam and/or thoracic X-ray. Besides, the early detection of pulmonary congestion at bedside is essential to optimize the treatment and improve the prognosis of unstable patients with decompensated HF.[41] The detection of B lines is originated due to an artifact caused by the presence of extravasated fluid in the interstitium and pulmonary alveoli, reducing lung aeration. Therefore, other pathologies that lead to this physiopathological process can originate the same ultrasound image (viral pneumonitis and ARDS, for instance). Therefore, there are some signals suggesting that B lines are caused by congestion: 1) concordance with echocardiographic parameters of increasing filling pressures in the LV or increased atrial pressure; 2) thin pleural line with preserved sliding; 3) absence of subpleural involvement; 4) bilateral distribution of B lines, with prevalence in basal pulmonary segments; 5) coalescent B lines and absence of spared areas; 6) coexisting pleural effusion.[41]

Besides making the diagnosis of pulmonary involvement and its probable etiology, it is essential to quantify the level of B lines identified in PUS, monitoring the treatment and helping care management in a serial manner.

There are several ways described in the literature for that quantification.[41] The most practical way is to add the number of B lines visualized in the full sweep: 6 to 15 – mild; 16 to 30 – moderate; > 30 – major. Another validated way is to add up the score found in each studied zone, and the points would be discriminated as follows: 0 (absence or up to 3 B lines), 1 (> 3 B lines), 2 (coalescent B lines) and 3 (consolidation).

The quantification of B lines carried out by PUS is highly correlated with the findings of the computed tomography and in the evaluation by transpulmonary thermodilution.[42] The monitoring of pulmonary involvement and the serial bedside evaluation are useful tools in many situations, such as: 1) identifying the ideal moment for the weaning of invasive mechanical ventilation and as a predictor of a flawed extubation, 2) assessing the response after interventions, such as maneuvers of physical therapy recruitment, ideal positive end-expiratory pressure (PEEP) adjustment, therapeutic bronchoscopy, estimation of volemia and response after diuretic therapy and evolution of lung aeration after devices of pulmonary circulation assistance.

PUS is also accurate to diagnose other conditions associated with critical patients (pneumothorax, pleural effusion, pneumonia and pulmonary embolism). In Figure 5, we describe the normal pattern and the images found in several pathological situations.

Some algorithms were proposed in the literature to guide the diagnosis based on PUS.[43] We can emphasize the modified BLUE and FALLS protocols (Figure 6), both with excellent accuracy and an important tool in the etiological investigation of patients with respiratory failure and circulatory shock, respectively.

Limitations of the PUS

Even though the method is easier to execute than other modalities of ultrasound, it requires training and development of skills; its learning curve is relatively short.

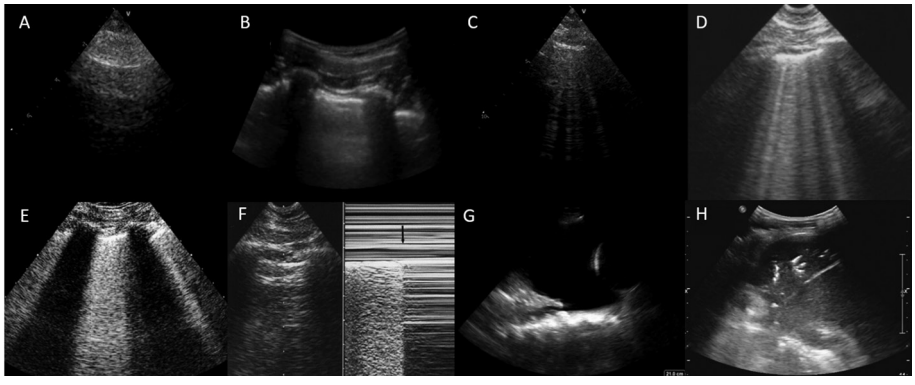

Figure 5 – *Normal and pathological findings in pulmonar ultrasound. Normal pulmonary ultrasound using a sector transducer (A) and a convex transducer (B). C. B lines in small quantity. D. B lines in large quantity with spared areas. E. B lines in large coalescent quantities. F. To the left, there are A lines in 2D and, to the right, the typical pattern of pneumothorax M-mode (arrow demonstrating lung point). G. Pleural effusion. H. Pneumonia.*

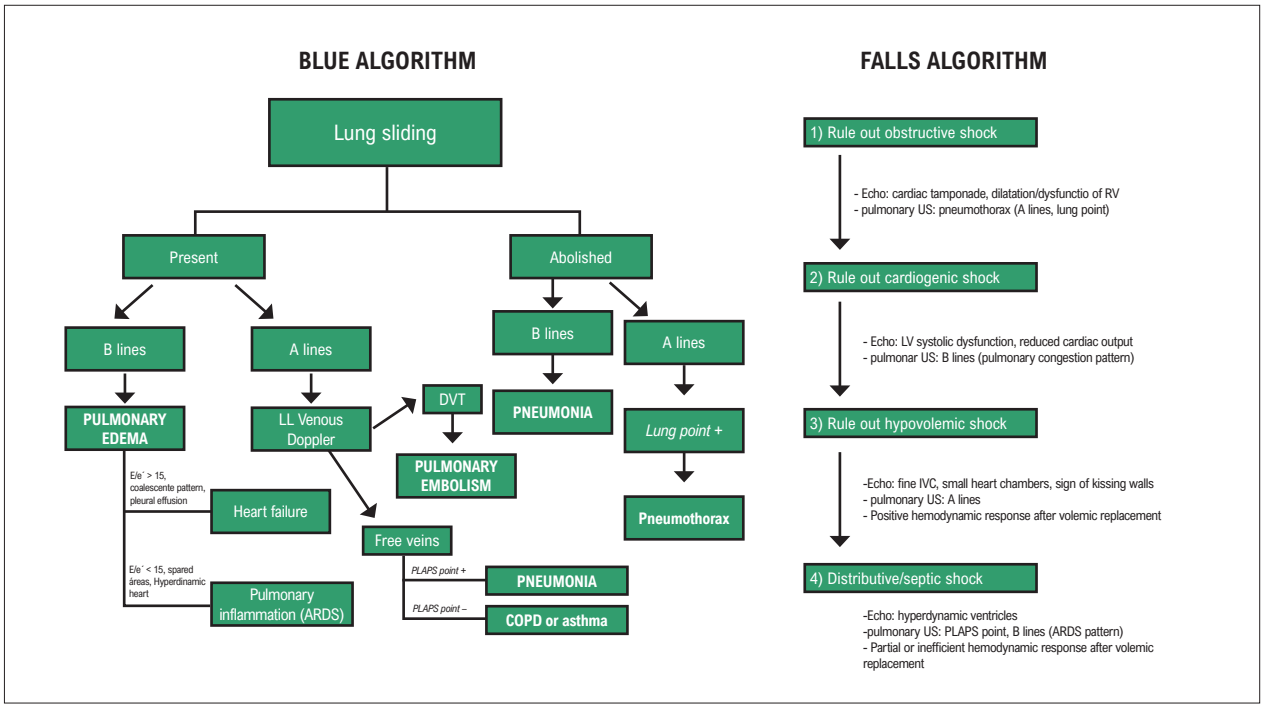

Figure 6 – *LL: lower limbs; ARDS: Acute respiratory distress syndrome; DVT: deep venous thrombosis; PLAPS: posterolateral alveolar and/or pleural syndrome; COPD: chronic obstructive pulmonar disease; RV: right ventricle; LV: left ventricle; IVC: inferior vena cava. Source: Adapted from Lichtenstein DA (2015).*[43]

The inter and intra-observer variability is small (lower than 5%), and the feasibility of the method is high. However, there may be difficulties related to the critical patient, such as inadequate acoustic window, difficulties in positioning, chest seals and/or presence of subcutaneous emphysema.[38]

PUS is very accurate to identify lung involvement and other comorbidities related to the critical patient. However, the findings should be interpreted within the context of each patient in the ICU, including clinical, laboratory parameters and other imaging exams.

Conclusions

The echocardiogram plays an essential role in the management of the patient in circulatory shock. It is a crucial help in etiological diagnosis, prognosis, hemodynamic monitoring and volemic estimation of these patients. Its application in a critical patient environment should be more and more incorporated to the daily practice.

Added to the strategy of bedside echocardiogram, the use of lung ultrasound and VExUS score may add relevant data to the estimation of volemia in a critical patient, thus influencing the probabilistic decision of fluid-responsiveness and aggregating information to the diagnostic logic of the causes of shock.

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Conception and design of the research and writing of the manuscript: Fernandes RM, Souza AC, Leite BF, Kawaoka JR; critical revision of the manuscript for intellectual content: Fernandes RM.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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